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Release of nitric oxide by activation of nonadrenergic noncholinergic neurons of internal anal sphincter.

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The purpose of the present study was to investigate the direct release of nitric oxide (NO) in response to the stimulation of nonadrenergic noncholinergic (NANC) nerves. The studies were performed on isolated smooth muscle strip of the opossum (Didelphis virginiana) internal anal sphincter (IAS). Electrical field stimulation (EFS) using the appropriate parameters caused a frequency-dependent fall in the resting tone of the IAS. The release of NO was measured directly by the chemiluminescence method. The stimulation of NANC neurons by EFS and the nicotinic stimulant 1,1-dimethyl-4-phenylpiperazinium (DMPP) caused IAS relaxation with an accompanying release of NO. The release of NO and the fall in the resting tension of IAS in response to lower frequencies of EFS and DMPP were abolished by pretreatment of the smooth muscles with the neurotoxin tetrodotoxin and the NO-synthase inhibitor NG-nitro-L-arginine (L-NNA). The obliteration of the release of NO and the IAS relaxation in the presence of L-NNA reversed to control levels by the addition of the NO precursor L-arginine. The effect of L-NNA and L-arginine on NO release and IAS relaxation was stereoselective, since D-NNA and D-arginine had no significant effect. Vasoactive intestinal polypeptide also caused release of NO from IAS smooth muscle strips, which was abolished by L-NNA. However, isoproterenol and atrial natriuretic factor caused IAS relaxation without any increase in NO release. In conclusion, these studies demonstrate the direct release of NO in response to the stimulation of NANC inhibitory neurons of the gut.

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